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The general goal of the ONR project was to determine the neural basis of learning and memory, that is, how the brain stores and retrieves memory. The special form of learning which was the focus of this project was conditioned taste aversions, learned aversions to the taste of food or fluid when consumption of that substance is followed by illness. Studies were made of the illness pathway and the illness-taste integration pathway. The conditions under which endogenous substances act as illness-inducing agents were determined, techniques to study the neural substrates for those substances as well as exogenous toxins were developed and evidence refuting hypotheses regarding the role of particular brain areas as substrates for illness-integration was obtained. In addition, endogenous factors that modulate the acquisition and extinction of conditioned taste aversions were identified. Variations in endogenous hormone levels, availability of water, and age alter the facility with which an aversion is learned and unlearned. Finally, a neural model encompassing the illness pathway, the taste pathway, the behavioral pathways, and the modulatory pathways was developed.

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GOALS

The general goal of the ONR project is to determine the neural basis of learning and memory, i.e., how the brain stores and retrieves memory. More specifically we are determining how the hard-wired (innate) part of the neural system interfaces with the plastic (learned) part. The special form of learning which is the focus of this project is conditioned taste aversions (CTAs), i.e., learned aversions to the taste of a food or fluid when consumption of that substance is followed by illness. In order to achieve this general goal, neurobiological and computational analyses of the neural network essentials for CTA are being integrated. The essential neurobiological network for CTA is being identified and characterized and computational models for the CTA neural circuit are being developed.

NEUROBIOLOGICAL RESEARCH

It is necessary to identify four pathways in order to gain a clear understanding of the neural basis of CTAs: the US (illness) pathway, the CS (taste) pathway, the pathway for the elicited response to the CS prior to conditioning (UR_{cs} or unconditioned ingestive response, UIR), and the pathway for the elicited response to the CS after conditioning (CR_{cs} or unconditioned aversive response, UAR). As much work has already been done on the taste and UR_{cs} pathways, we are concentrating on the illness and illness-taste integration pathways in this proposal. In addition, we also are identifying endogenous factors that modulate the acquisition and extinction of conditioned taste aversions.

Illness Pathway

There are two known detection systems for toxins, the gastric-intestinal mucosa and the area postrema. The vagus nerve conveys information from the gastric-intestinal mucosa to the nucleus of the solitary tract (NST), pontine parabrachial nucleus

(PBN) and the insular cortex (Cechetto & Saper, 1987; Norgren, 1978; Torvik, 1956). The area postrema detects chemicals in the blood and is thought to convey this information to the NST (Morest, 1957). Beyond this little is known about the illness pathways.

A wide variety of substances can induce CTAs. The detection system that is used to convey information about these substances to the brain varies with the particular chemical and the route of administration. LiCl, a widely used illness-inducing agent, acts by way of the area postrema; copper sulfate acts by way of the vagus nerve when it is administered intraperitoneally; and, apomorphine acts by systems other than the area postrema and the vagus nerve.

One of our aims is to determine the conditions under which endogenous substances act as illness-inducing agents in a CTA and to determine the neural pathways by which these agents and the commonly found toxin, LiCl, produce their effect.

Experimental Series 1: Nature of Toxin

We have completed 8 experiments aimed at determining the role of estradiol in conditioned taste aversions and its possible toxic effects (Experiments 1-8). One of these experiments was included in a paper that has been published in Physiology and Behavior and data from the rest of the experiments were presented at the American Psychological Society meetings in 1989, 1990, and 1991.

We have demonstrated that estradiol prevents testosterone from prolonging extinction of a conditioned taste aversion when it is administered during acquisition or during extinction (Experiment 1; Chambers & Yuan, 1990; Yuan & Chambers, APS-1989). Since testosterone is effective in slowing extinction only when it is present during extinction, estradiol does not have to be present concurrently with testosterone during extinction to be effective. This suggests that estradiol does not act on a testosterone-related mechanism but rather acts independently of testosterone.

If estradiol acts independently of testosterone, then one would expect that estradiol would increase extinction rate regardless of the presence of testosterone during extinction. We have found that when only estradiol is given during acquisition and extinction of a conditioned taste aversion, extinction is accelerated (Experiment 2; Yuan & Chambers, APS-1989). This effect is found in both females and males (Experiment 3) and in Fischer 344 and Sprague-Dawley rats (Experiment 4) and it is found under different doses of LiCl (Experiment 5). This effect is not found, however, in aged females (Experiment 6; Yuan & Chambers, APS-1990). This suggests that aged females have a reduced sensitivity to estradiol.

In a recent study, we obtained further evidence to support the hypothesis that estradiol and testosterone act by way of separate mechanisms (Experiment 7; Yuan & Chambers, APS-1991). We have found that estradiol accelerates extinction by acting on processes associated with acquisition not extinction. Testosterone, on the other hand, acts on processes associated with extinction not acquisition. The presence of estradiol around the time of acquisition accelerates extinction but when it is present around the time of extinction it has no effect.

Estradiol is known to produce nausea and vomiting in humans. Recently, it has been reported in other labs that increases in estradiol can induce the development of learned food aversions to available foods (Bernstein & Fenner, 1983). This effect is attenuated after lesions of the area postrema (Bernstein et al., 1985, 1986). We have found that estradiol accelerates extinction of an aversion and prevents testosterone from prolonging extinction. It is possible that estradiol produces these effects because of its toxicity. Preexposure to a toxin before acquisition can attenuate the subsequent learning of an aversion and preexposure to a toxin after acquisition but before extinction of an aversion can accelerate the subsequent extinction of the aversion. The putative explanation for this effect is as follows. Animals do not readily acquire aversions to familiar foods, expecially foods eaten from infancy. Thus when an animal is exposed to a toxin and only familiar foods are available, a dissassociation between illness induced by the toxin and food occurs. When a novel food is made available, this dissassociation then interferes with the ability of an animal to associate the illness with the novel food. One prediction that can be made on the basis of this explanation is that when the preexposure toxin is given before acquisition or before extinction, the aversion will be attenuated but when the preexposure toxin is given during acquisition and during extinction, the aversion will be strengthened. It may be that E acts as a preexposure toxin and that the effects of E during extinction cancelled out its opposite effects before extinction. In Experiment 8, we found that estradiol prolonged extinction when it was present during extinction but it had no effect on extinction when it was present prior to extinction (Yuan & Chambers, APS-1991). These results are consistent with the hypothesis that the effect of estradiol on the extinction of a conditioned food aversion is toxic related.

Experimental Series 2: Neural Pathways

We decided to concentrate our efforts on the role of the area postrema in mediating the effects of estradiol and other toxins such as LiCl. Thus we put aside pursuing the 2-DG studies and the multi-unit recording studies to identify active areas of the brain after administration of illness-inducing agents. Instead, we have developed a temporary lesioning technique whereby the area postrema is cooled during estradiol administration. The study to determine whether cooling the area

postrema blocks the effects of estradiol and LiCl is currently underway.

Illness-Taste Integration Pathway

We have completed 4 experiments aimed at identifying neural areas mediating illness-taste integration (Experiments 9-12).

Experimental Series 1: Role of the Amygdala

Several brain structures have been implicated in CTAs, but until recently, lesions of the amygdala (AMG), in particular the basolateral AMG, have produced the most consistent findings; they disrupt acquisition and retention of prelesion CTAs (Aggleton et al., 1981; Nachman & Ashe, 1974; Simbayi et al., 1986). But after finding that cutting the connections between the AMG and the temporal cortex produced the same deficits as lesions of the basolateral AMG, Fitzgerald and Burton (1983) suggested that it is the destruction of the fibers of passage that produces the deficits after lesions of the basolateral AMG and not the destruction of the nucleus itself. Recently, Dunn and Everitt (1988) found that neurotoxic (ibotenic)-induced lesions which spare the fibers of passage had no effect on aversion learning whereas electrolytic (ELEC) lesions which destroy both cells and fibers attenuated the aversion. Neither ibotenic or ELEC lesions had a significant effect on extinction. They concluded that it is the axons passing between the brain stem/hypothalamus and GN that are responsible for the deficits in acquisition after ELEC lesions of the AMG. In Experiments 9 and 10, we set out to examine the effect of ELEC and neurotoxic (NMDA, N-methyl-D,L-aspartic acid)-induced lesions on acquisition and extinction of a CTA when lesions were made before acquisition (Experiment 9) or after acquisition (Experiment 10).

We have completed all behavioral testing and histological examination for the two experiments. Acquisition was attenuated in rats with ELEC lesions but not NMDA lesions when lesions were made before acquisition or after acquisition (Chambers, 1990). Neither ELEC or NMDA lesions affected extinction. These results suggest that the basolateral AMG does not play a role in acquisition or extinction of conditioned taste aversions.

Experimental Series 2: Role of the Gustatory Cortex

Animals with lesions of the gustatory cortex (GN) exhibit slower acquisition of CTAs (Braun et al., 1972) and no retention of a prelesion CTA (Braun et al., 1981; Kiefer et al., 1984; Yamamoto et al., 1980). The effect of GN lesions on extinction is unclear. We have completed two studies designed to determine the effect of GN lesions on acquisition of a CTA (Experiments 11 & 12). In Experiment 11, the lesion was made before acquisition of a CTA and in the Experiment 12, lesions were made after

acquisition. In both experiments, male rats were randomly divided into 2 groups: sham control and lesion (by aspiration). The results from both experiments indicate that there is no effect of GN lesions on acquisition of a CTA. As these results differ from previous reports, an analysis of differences between those studies and ours will be made. Some obvious differences include the extent of the lesion, strain of rat, and fluid deprivation state.

Modulating Factors

We have completed 21 experiments aimed at identifying factors that modulate CTAs (Experiments 13-33). Two of these experiments have been combined in a paper that has been published in Behavioral Neuroscience and two other experiments have been combined in a paper that has been submitted to Neurobiology of Aging. Other experiments have been or will be presented at the following meetings: Society for Neuroscience, 1988, 1990, 1991; Third IBRO World Congress of Neuroscience, 1991; Western Psychological Association, 1989; American Psychological Society, 1990; The Gerontological Society of America, 1990, 1991.

Experimental Series 1: Hormonal Effects on Conditioned Taste Aversions

Effects of perinatal testosterone on extinction of conditioned taste aversions. We have found that the rate of extinction of a CTA in rats is dependent on concurrent levels of testosterone. When testosterone is administered to gonadectomized males and females, extinction is prolonged. However, females are less sensitive to testosterone than males. We have found that it is the presence of testosterone during the perinatal period that alters sensitivity to testosterone. Adult gonadectomized rats that had low levels of testosterone present during the perinatal period (normal females) exhibited a fast rate of extinction when given a low dose of testosterone whereas adult gonadectomized rats that had testosterone present during the perinatal period (males and androgenized females) showed a slow extinction rate when given the same low dose. Thus, although the presence of testosterone during the perinatal period is not critical for the expression of a slow extinction rate, it does reduce the amount of testosterone required to produce the slow rate (Experiment 13; Sengstake & Chambers, 1990).

Role of the Medial Preoptic Area in the Modulation of Testosterone. Testosterone has been shown to prolong the extinction rate of a conditioned food aversion in the rat. The neural substrate that mediates this effect has not yet been identified. The medial preoptic area has been shown to have high concentrations of androgen receptors and to play a crucial role in many androgen-mediated behaviors. Experiment 14 was designed

to determine whether the medial preoptic area is involved in the effect of testosterone on conditioned food aversions. The extinction rates of gonadectomized male rats with implants of testosterone in the medial preoptic area were compared to those of control rats. The testosterone-treated males had significantly slower extinction rates than the control males. These results support the hypothesis that the prolonged extinction induced by testosterone can be mediated by the medial preoptic area (Hung, Yuan & Chambers, IBRO-1991).

Effects of gonadectomy on extinction of a conditioned food aversion. Gonadal hormones alter the rates of extinction of conditioned food aversions in rats. Males have slower extinction rates than females. Gonadectomy increases the rates in males but has no effect in females. Testosterone treatment slows extinction in both males and females. We have observed that gonadectomy increases the extinction rates of males to those of females in Sprague-Dawley but not Fischer 344 rats (Experiment 15). In studies of reproductive behavior, the decrease in sexual activity after gonadectomy occurs over a long period of time. One possible explanation for the difference in the effects of gonadectomy in Sprague-Dawley and Fischer 344 rats is that gonadectomy may take longer to show an effect in Fischer 344. Experiments 16 and 17 were designed to determine whether the extinction rates of Fischer 344 rats varies with the length of time after gonadectomy. Males that were gonadectomized before puberty and given a conditioned food aversion 8 weeks later (Experiment 17) and males that were gonadectomized after puberty and given a conditioned food aversion 5 weeks later (Experiment 16; Yuan, Hung & Chambers, WPA-1989) showed a faster extinction rate than males gonadectomized after puberty and given a conditioned food aversion 1 week later. However, all gonadectomized males still extinguished more slowly than females. These results suggest that differences in the effects of gonadectomy on extinction rates in Sprague-Dawley and Fischer 344 rats may be accounted for, at least in part, by differences in the length of time the effects of gonadectomy are expressed.

Experimental Series 2: Age-related changes in acquisition and extinction of conditioned taste aversions in males

There is an age-related decrease in the sensitivity of the target tissues mediating sexual behavior to testosterone. The following experiments were designed to determine whether the same was true for target tissues mediating testosterone modulation of CTAs. Some investigators have reported prolonged extinction which is opposite of what one would expect in aged males with decreased testosterone levels. Thus in Experiment 18, the extinction rates of 10 young (3 months) and 10 old (18 months) males were examined. The old males had slower extinction rates (Yuan, Hung & Chambers, APA-1990). This age-related difference was eliminated by gonadectomizing the old males (Experiment 19).

In Experiment 20, the acquisition rates of 40 young (3 months) and 40 old (16 months) males were examined. The young and old males were given 4 different doses of illness-inducing toxin. An analysis of the results indicates that old males do not acquire a CTA as readily as young males when low doses of LiCl are used. For those old and young males that acquired an aversion at a given dose, no differences in acquisition rate or extinction rate were found. These results suggest that old males have a lower sensitivity to LiCl than young males and that they differ from young males in how they process information about LiCl when one high dose is given but not when repeated low doses are given (Yuan, Hung & Chambers, APA-1990, GSA-1990). The reduced sensitivity to LiCl observed in old males can be reversed by testosterone treatment (Experiment 21; Yuan, Diego & Chambers, GSA-1991). This suggests that the decreased sensitivity to LiCl in old males is due to their decreased testosterone levels.

Experimental Series 3: Fluid Deprivation Effects on Conditioned Taste Aversions

The rate of extinction of a conditioned taste aversion is dependent on testosterone levels; males with normal physiological levels exhibit slow extinction and gonadectomized males show fast extinction. Fluid deprivation also increases the rate of extinction in male rats. Studies were done to determine the relationship between fluid deprivation and testosterone. In Experiment 22, we found that testosterone levels are lower in Sprague-Dawley fluid deprived males than in nondeprived males (Brownson, Sengstake & Chambers, SN-1988). Testosterone treatment slowed extinction rate in fluid deprived males but the amount of testosterone needed to slow extinction was greater than the amount needed to restore testosterone blood levels to normal (Experiment 23). These results suggest that fluid deprivation decreases sensitivity to testosterone.

In experiments with Fischer 344 fluid deprived males we observed that fluid deprivation did not have the same effects as found in Sprague-Dawley fluid deprived males. Fluid deprivation does not decrease testosterone levels in fluid deprived Fischer 344 males (Experiment 24). Also, the percentage increase in the extinction rates of fluid deprived Fischer 344 males is greater than that of fluid deprived Sprague-Dawley males (Experiment 25, Brownson, Sengstake & Chambers, SN-1990).

The ability of testosterone to slow extinction in fluid deprived Fischer 344 male rats depends on the dose of LiCl used and on whether the male is gonadectomized or intact. Testosterone replacement was less effective in gonadectomized males than intact males (Experiment 26, 27 & 28). Testosterone was more effective in slowing extinction in fluid deprived Fischer 344 males administered a high dose of LiCl (0.30 M, 10 ml/kg) than a lower dose (0.15 M, 10 ml/kg; Experiment 28 & 29).

Fluid deprivation also increases extinction rate in female rats (Experiment 30 & 31). The increased extinction rate found in females and males cannot be accounted for by the fact that the animals are thirsty. When fluid deprived males and females are allowed to drink water for 10 min before being given access to sucrose, they still show a fast extinction (Experiment 32 & 33).

All of these results suggest that fluid deprivation does not act on a testosterone mechanism to increase extinction rate.

COMPUTATIONAL RESEARCH

We have completed a paper that lays the groundwork for developing a computational model for CTAs (Chambers, 1990).

The determination of the neural substrates for CTAs should involve the identification of four pathways: the US pathway, the CS pathway, the pathway for the elicited response to the CS prior to conditioning (UR_{cs} or unconditioned ingestive response, UIR), and the pathway for the elicited response to the CS after conditioning (CR_{cs} or unconditioned aversive response, UAR). Each taste is connected to both the ingestive and aversive patterns of responses. These connections are probably innate as hedonic reactions to taste have been observed in fetal and neonatal individuals (Pfaffman 1978, Steiner 1973, 1979).

The relative strengths of the two innate connections are dependent on the given taste. In the case of sucrose, the innate connection to the ingestive response is stronger than the innate connection to the aversive response. If exposure to sucrose is followed by illness, the connection to the ingestive response system will weaken and the connection to the aversive response system will strengthen. It is most likely that the illness-induced changes involve two rather than one process. Grill and Berridge (1985) have suggested that palatability processing involves two mechanisms and have provided evidence that the ingestive and aversive response systems can change independently. Thus, in order for the aversive response system to be expressed solely, a weakening of the ingestive response system would have to occur. If exposure to sucrose is not followed by negative consequences, a stronger connection to the ingestive response system will result. A stronger connection to the ingestive response system also will occur if a given taste is associated with positive reinforcement or if it is followed by recuperation from illness (Garcia et al 1977, Revusky 1967, 1974, Young 1966). So, experiential factors can alter the strengths of the innate connections to the ingestive and aversive response patterns. Thus, after a given taste is experienced, the relative strengths of the ingestive and aversive response systems are a function of the original innate connections, the number of exposures to sucrose with illness and the number of exposures to sucrose without illness. This hypothesis is supported by the findings that CTAs to nonpreferred tastes are stronger than to preferred tastes (Etscorn 1973), repeated pairings of a taste with illness

strengthens an aversion and repeated pairings of a taste without illness reduces the strength of an aversion (Kalat & Rozin 1973).

There are other factors associated with the CS and US that can influence the strength of an aversion and therefore must be taken into account when developing a neural model for CTAs. The strength of an aversion has been found to be a function of the intensity of the taste as measured by concentration (Dragoin 1971) and the amount consumed on the first exposure (Bond & DiGuisto 1975), the intensity of the US (Revusky 1968) and prior experience with the US (Cannon et al 1975).

There are several factors which can modulate the development and strength of CTAs, but are not essential or critical for aversion learning. The development and strength of an aversion is dependent on the hormonal milieu and deprivation state of the animal. The presence of testosterone (T) increases the proportion of animals that develop a CTA (Chambers et al 1981) and the presence of dexamethasone attenuates the strength of an aversion (Hennessy et al 1976). Water deprivation reduces the proportion of male rats that develop an aversion (Chambers et al 1981). It is interesting that deprivation can alter the hedonic value of tastes. Foods are reported to be more palatable with deprivation and less so with satiety (Cabanac 1971). Also, the number of ingestive responses decreases and the number of aversive responses increases as meal termination approaches (Grill & Berridge 1985). So, the relative strengths of the ingestive and aversive response systems are also a function of modulating factors. A complete understanding of the neural mechanisms controlling CTAs would include a determination of the neural circuitry for the modulating factors.

A neural model for extinction of a CTA can be outlined in a similar manner as acquisition. Extinction is a process by which connections to the aversive response system are weakened and connections to the ingestive response system are strengthened. Any information on the subsequent consequences of ingesting the CS is processed. If the consequences are neutral, that information serves to alter the relative strengths of the two response systems. Thus, after a CS has been experienced without negative consequences, the relative strengths of the ingestive and aversive response systems are a function of the relative strengths of these systems after the CTA, the number of exposures to the taste without illness, modulating factors, and probably the original innate predisposition.

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Chambers, K. C. A neural model for conditioned taste aversions; Annual Review of Neuroscience, 13, 373-385, 1990.

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MANUSCRIPTS IN PREPARATION

Brownson, E. A., Sengstake, C. B., and Chambers, K. C. Strain differences in the effects of fluid deprivation of testosterone levels.

Chambers K. C., Brownson, E. A., and Sengstake, C. B. Effects of fluid deprivation on testosterone levels and modulation of extinction of conditioned taste aversions.

Yuan, D. and Chambers, K. C. Effects of estradiol on extinction of conditioned taste aversions.

Yuan, D. and Chambers, K. C. Changes in acquisition and extinction of conditioned taste aversions in aging male rats.

PRESENTATIONS AT MEETINGS

Yuan, D. and Chambers, K. C. Temporal analysis of estradiol blockage of testosterone effect on conditioned taste aversions. Poster presented at the first annual meeting of the American Psychological Society, Alexandria, VA, June 1989.

Brownson, E. A., Sengstake, C. B., and Chambers, K. C. Effects of fluid deprivation on testosterone sensitivity and extinction of a conditioned taste aversion. Poster presented at the 20th annual meeting of the Society for Neuroscience, St. Louis, 1990.

Yuan, D. L. and Chambers, K. C. Age-related difference in the effect of estradiol on extinction of a conditioned food aversion. Poster presented at the second annual meeting of the American Psychological Society, Dalles, TX, June 1990.

Yuan, D. L., Hung, C. and Chambers, K. C. Effects of gonadectomy on extinction of a conditioned food aversion. Abstract submitted for the Western Psychological Society meeting, Los Angeles, CA, April 1990.

Yuan, D. L., Hung, C. and Chambers, K. C. Conditioned food aversion in young and old male rats. Poster presented at the American Psychological Association meeting, Boston, MA, August 1990.

Yuan, D. L., Hung, C. and Chambers, K. C. Failure to observe age-related differences in extinction of a conditioned food aversion when low doses of LiCl are used. Poster presented at the Gerontological Society meeting, Boston, MA, November 1990.

PRESENTATION REQUESTS ACCEPTED

Yuan, D. L. and Chambers, K. C. Condition under which estradiol prolongs extinction of conditioned food aversion. Poster to be presented at the third annual meeting of the American Psychological Society, Washington, D.C., 1991.

PRESENTATION REQUESTS SUBMITTED

Hung, C., Yuan, D. L., and Chambers, K. C. Medial preoptic implant of testosterone can prolong the extinction of a conditioned food aversion in the male rat. Submitted to the Third IBRO World Congress of Neuroscience, Montreal, Canada, August, 1991.

Brownson, E. A., Sengstake, C. B., and Chambers, K. C. Effectiveness of testosterone in prolonging extinction of conditioned taste aversions in fluid deprived male rats depends on LiCl dose. Submitted to the Society for Neuroscience, New Orleans, November, 1991.

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